SCIENTIFIC LETTER

Mid systolic septal deceleration in hypertrophic cardiomyopathy: clinical value and insights into the pathophysiology of outflow tract obstruction by tissue Doppler echocardiography

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Hypertrophic cardiomyopathy (HCM) is an inherited cardiac disorder characterised by abnormal left ventricular (LV) hypertrophy in the absence of severe aortic stenosis and hypertension. The presence of a dynamic gradient in the left ventricular outflow tract (LVOT) above 30 mm Hg at rest is associated with an increased mortality.[1] Surgical myectomy or transcoronary ablation of septal hypertrophy (TASH) is indicated in patients with persistent symptoms and gradients > 50 mm Hg under resting or provoked conditions, despite medical treatment.[2] We recently described a characteristic mid systolic septal deceleration (MSSD) pattern in the velocity trace of the basal septum obtained by tissue Doppler imaging (TDI),[3] but the diagnostic value of the MSSD pattern in a larger patient population is still unknown.

PATIENTS AND METHODS

Twenty six HCM patients (15 male, mean (SD) age 48 (17) years, septal wall thickness > 15 mm) were studied by transthoracic resting echocardiography (GE-Vingmed Vivid 7 Pro, Horten, Norway). In addition to the standard measurements, we analysed longitudinal septal motion by colour coded TDI from the apical view (frame rate 192 (40)/s). All data were stored digitally and analysed off line (EchoPac, GE-Vingmed, Horten, Norway). The MSSD pattern was defined by the presence of a biphasic systolic velocity curve with an early systolic peak (S_1), followed by a sudden deceleration with a mid systolic nadir ($\geqslant 1$ cm/s and/or $\geqslant 25\%$ decrease from S_1) and a second systolic velocity peak (S_2) (fig 1). Paired data were analysed with Wilcoxon signed rank test, unpaired data were analysed with Mann-Whitney U test.

RESULTS

All patients showed normal systolic LV size and function (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm, fractional shortening 46 (10)%, and significant LV hypertrophy (interventricular septum 22 (5) mm; posterior wall 14 (3) mm). The average LVOT resting gradient was 49 (35) mm Hg. A clinical relevant LVOT resting gradient (> 30 mm Hg) was observed in 15/26 patients (58%) and in 14 of those associated with mitral systolic anterior motion (SAM) and mid systolic septal contact. An MSSD pattern was present in 15/26 patients (58%) and occurred in all patients simultaneous to the development of LVOT obstruction. The MSSD notch was associated with significant LVOT obstruction > 30 mm in 14/15 patients (93%). In one patient an MSSD notch was present despite a slightly lower LVOT gradient of 25 mm Hg. The presence of an MSSD pattern identified patients with LVOT

gradients above 30 mm Hg with 93% sensitivity and 91% specificity and was associated with higher LVOT gradients (71 (27) v 17 (10) mm Hg, p < 0.001), more septal hypertrophy (24 (2) mm v 19 (4) mm, p < 0.01), larger left atrial size (48 (7) mm v 41 (6) mm, p < 0.05) and smaller systolic LVOT area by planimetry (0.5 (0.3) mm^2 v 1.4 (1.2) mm^2).

DISCUSSION

The presence of an MSSD pattern is closely related to dynamic LVOT obstruction (fig 1). It resembles the classic spike and dome pattern as described in aortic pressure and carotid pulse tracings. Similar patterns have been described by conventional Doppler methodology.[4] Our TDI results demonstrate that the presence of LVOT obstruction is not limited to alterations in blood pressure and flow, but has also an impact on LV mechanical function.

However, the mechanisms responsible remain to be elucidated. The sudden deceleration of longitudinal septal motion might be caused by an external force, pushing or dragging the septum towards the base. However, this cannot be explained by the LVOT gradient itself, which develops in parallel to the septum. Alternatively, it might be caused by an internal unbalance of myocardial forces within the septum. Normal systolic radial and longitudinal myocardial thickening occurs almost simultaneously. Dynamic LVOT obstruction leads to a sudden rise in LV pressure during mid systole. The excessive rise in LV wall stress will impede further myocardial thickening in the radial direction and this will also hamper longitudinal shortening caused by the conservation of mass principle. The only segment which is not affected by the abnormally increased LV cavity pressure is the basal septal myocardial segment which lies below (downstream to) the region of mitral septal contact, thus being exposed to a lower mid systolic wall stress.[5] Isolated longitudinal shortening of this basal septal segment will drag the septum towards the base, thereby causing the MSSD notch. This abnormal septal motion may contribute further to LVOT narrowing and the positive amplifying feedback loop of LVOT obstruction.

The present study was limited to gradient evaluation at rest and did not systematically evaluate the influence of exercise on the LVOT gradient and on septal TDI patterns. Some patients may develop significant LVOT obstruction and an MSSD pattern during exercise despite a non-significant resting gradient. Thus, the absence of an MSSD pattern at

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Abbreviations: S_1, early systolic peak; HCM, hypertrophic cardiomyopathy; LV, left ventricular; LVOT, left ventricular outflow tract; MSSD, mid systolic septal deceleration; S_2, second systolic velocity peak; SAM, systolic anterior motion; TDI, tissue Doppler imaging
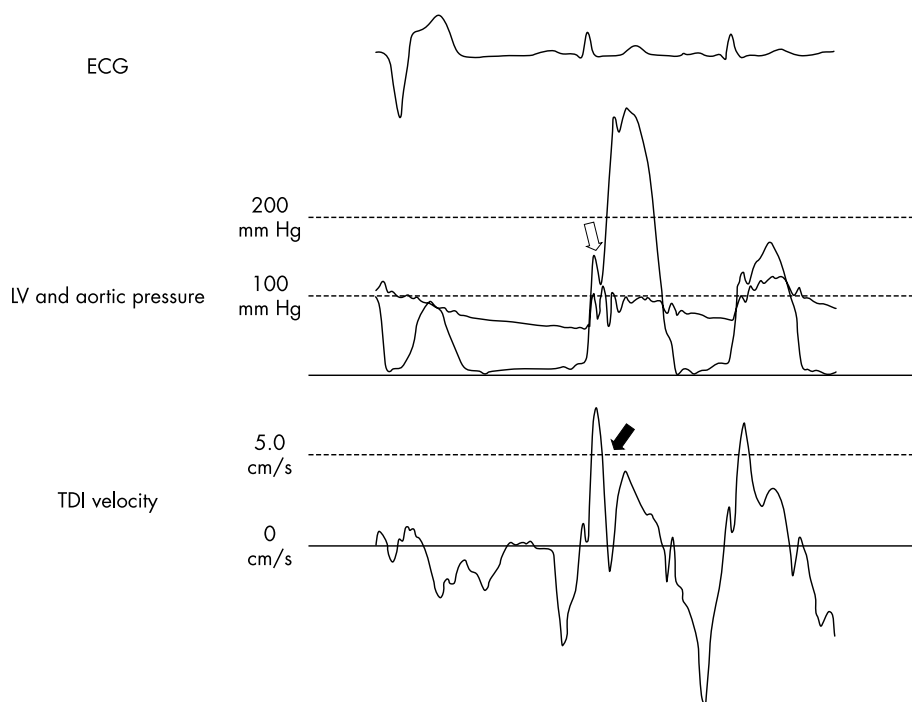
Figure 1 ECG invasively measured left ventricular outflow tract (LVOT) pressure gradient and the tissue Doppler imaging (TDI) velocity trace from the basal septum. Note the simultaneous development of the LVOT gradient (open arrow) and the mid systolic septal deceleration notch (solid arrow). Adapted from Breithardt and colleagues.[3]

rest does not exclude the presence of LVOT obstruction during exercise.

In conclusion, the presence of an MSSD pattern in the septal TDI velocity trace, defined and identified by the presence of two systolic velocity peaks and a sudden interpolated deceleration notch, identifies HCM patients with clinically important LVOT obstruction. TDI analysis of septal longitudinal motion patterns may constitute a new diagnostic tool additional to the conventional continuous wave Doppler examination for gradient measurement. It may help to verify the presence of an LVOT gradient, particularly in difficult imaging conditions such as exercise testing and in the presence of mitral regurgitation. This additional information is likely to reduce the number of false negative studies in patients where conventional Doppler methods fail to identify the site of gradient development, and it will identify false positive cases where the continuous wave Doppler beam has been misaligned and records mitral regurgitation instead of the LVOT velocity. Whether the presence or absence of an MSSD notch will improve risk stratification in HCM patients, or whether it may help to validate the response to treatment, remains to be studied prospectively.

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